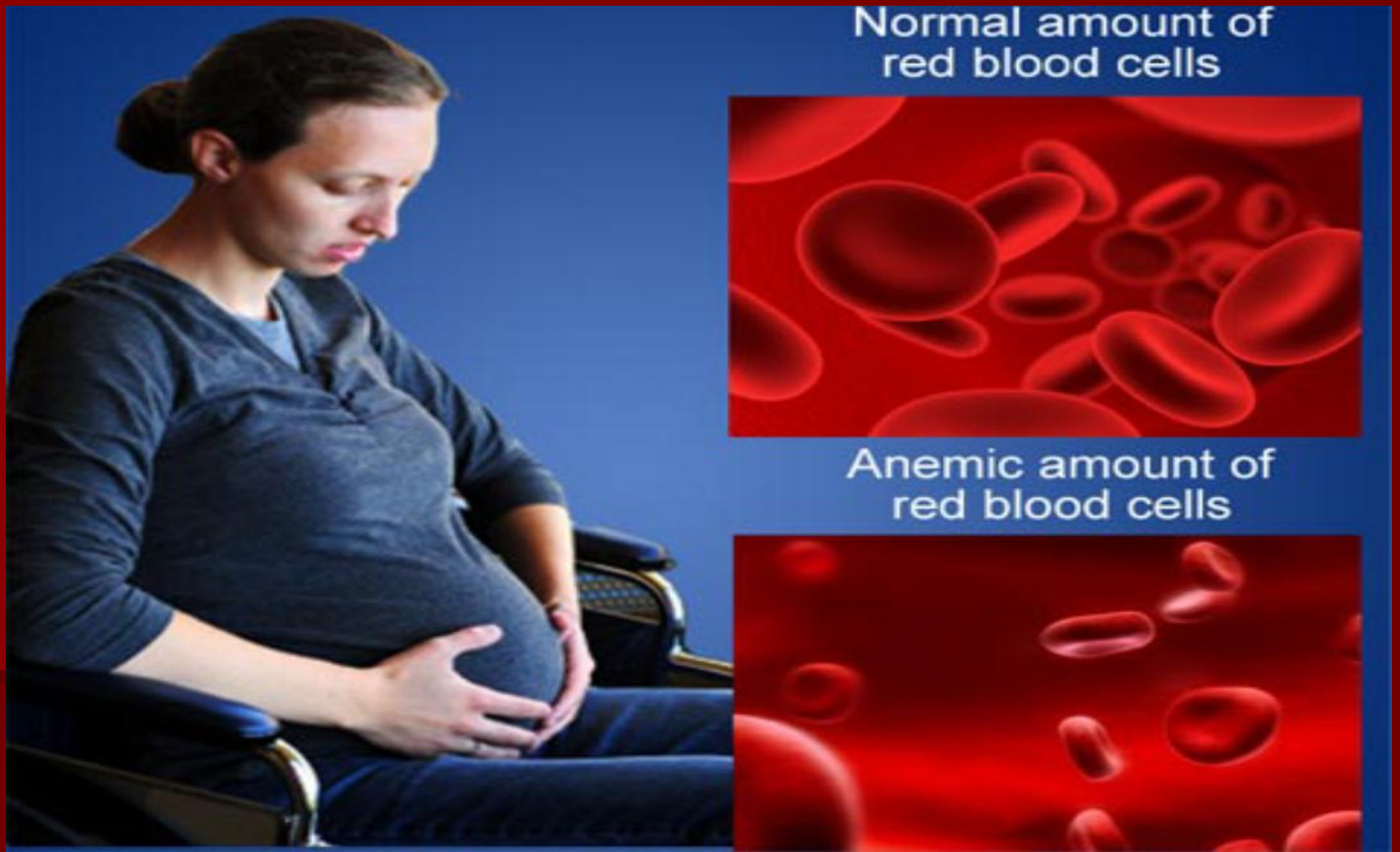
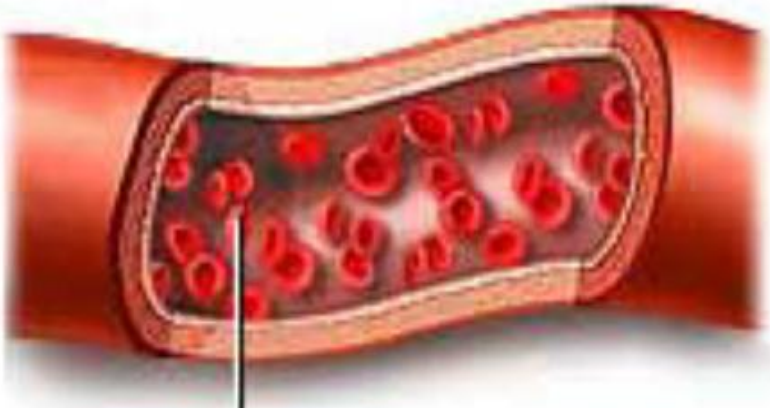
Anaemia in Pregnancy

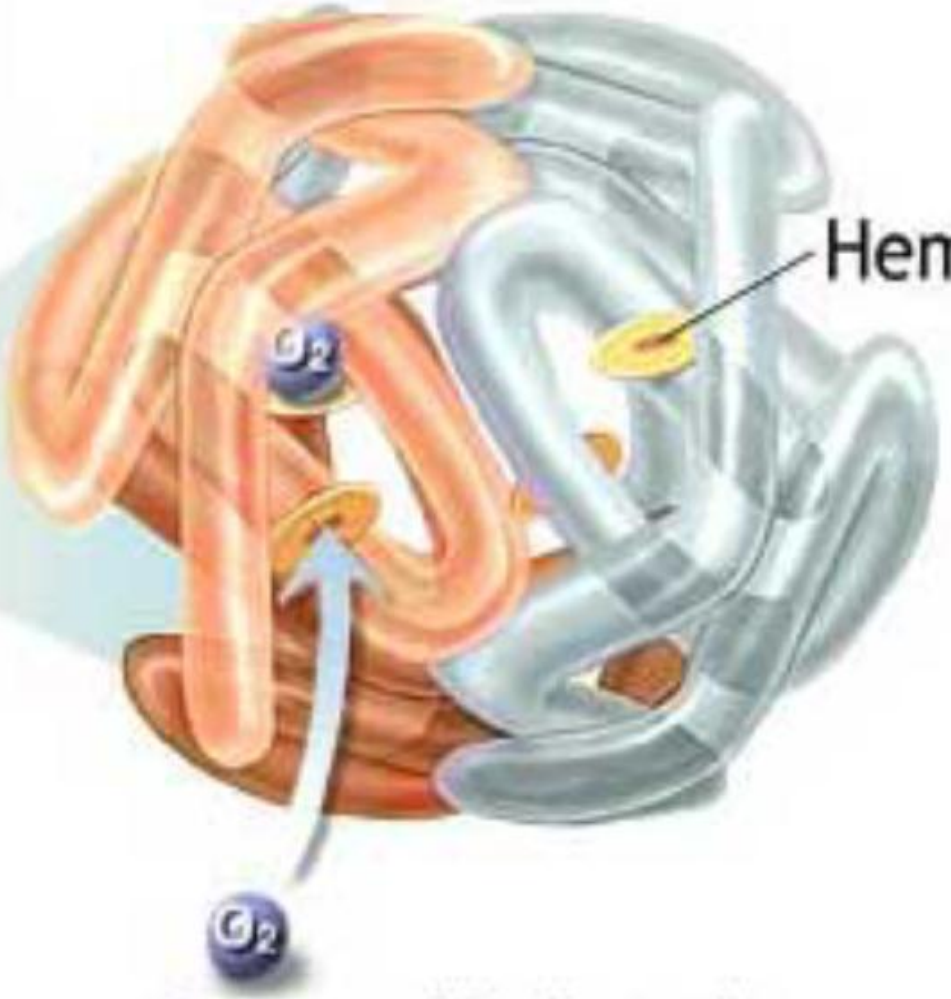




Red blood cell

Red blood cells contain several hundred hemoglobin molecules which transport oxygen

Hemoglobin molecule



Oxygen binds to heme on the hemoglobin molecule

Definition :anaemia is a pathological condition in which the oxygen-carrying capacity of red blood cells is insufficient to meet the body needs .

The WHO : haemoglobin concentration of < 11.0 g/dL in the 1st trimester & < 10.5 g/dL in 2nd & 3rd trimesters.

Incidence:

30-50% of women become anaemic during pregnancy,

Iron deficiency anaemia responsible for more than 90% of cases.

Folate deficiency is 5%.

Screening during pregnancy

Severity of Anemia:

Mild : Hb 10-10.9 g/dL

Moderate : Hb 7-9.9 g/dL

Severe :4-6.9 g/dL

Very sever : < 4 g/dL

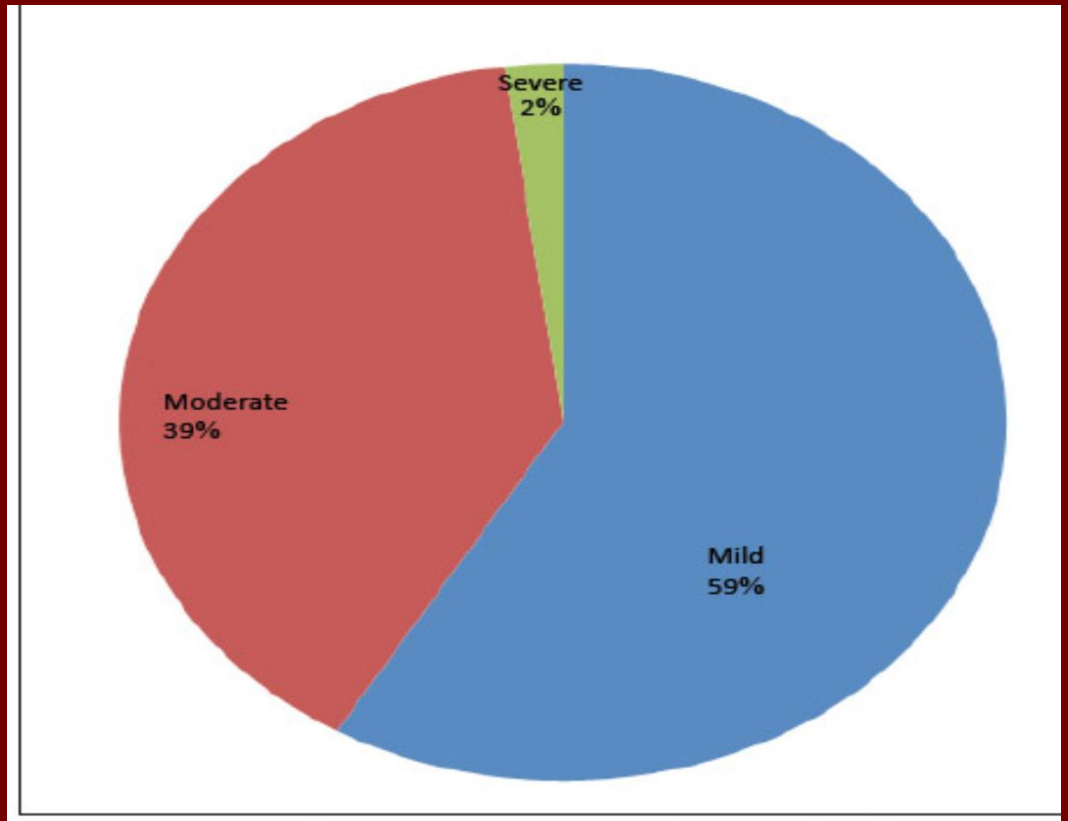
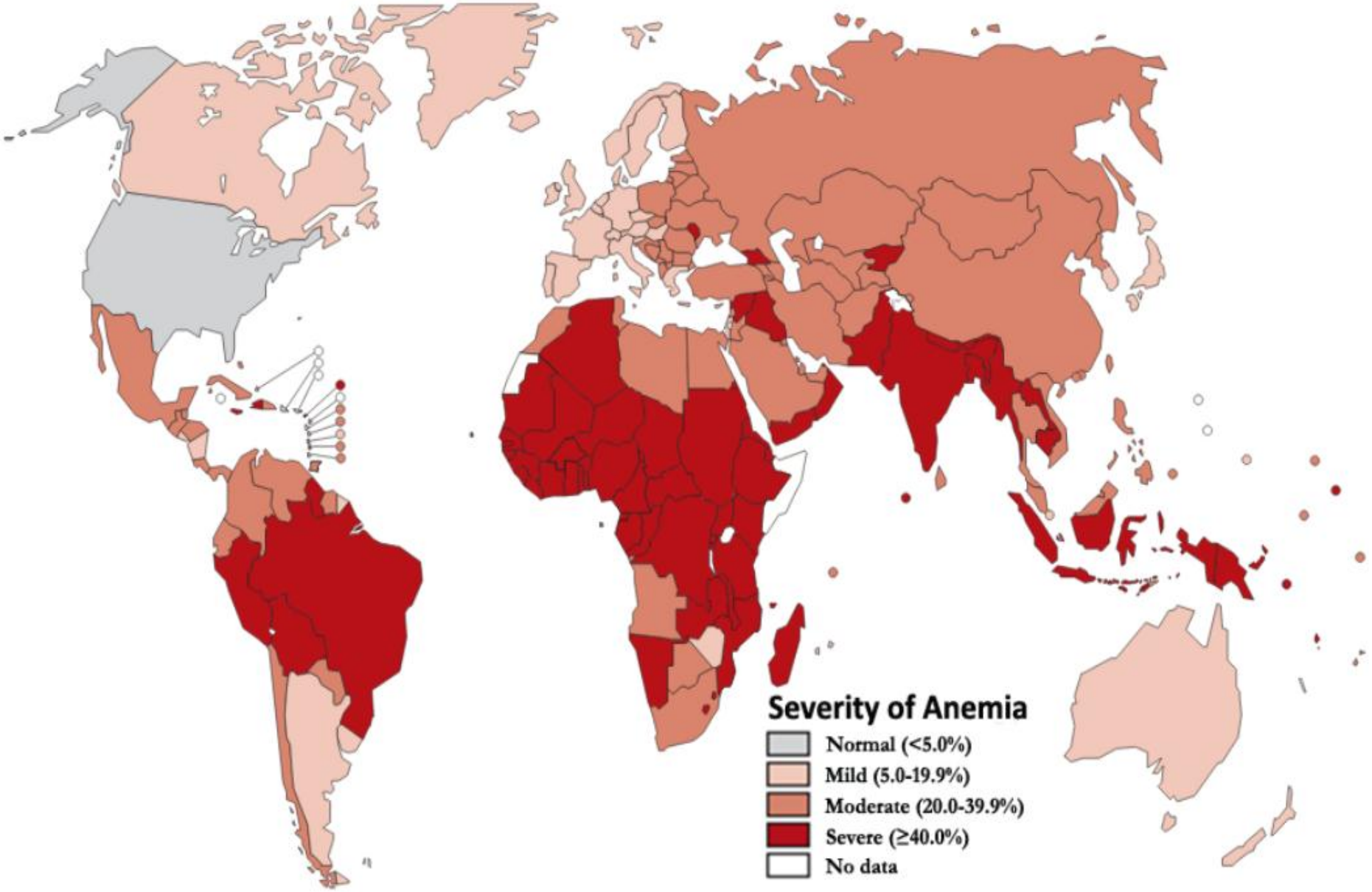


FIGURE 2: Severity of anaemia in pregnancy in the primary health centre.

Worldwide Prevalence of Anemia, by severity



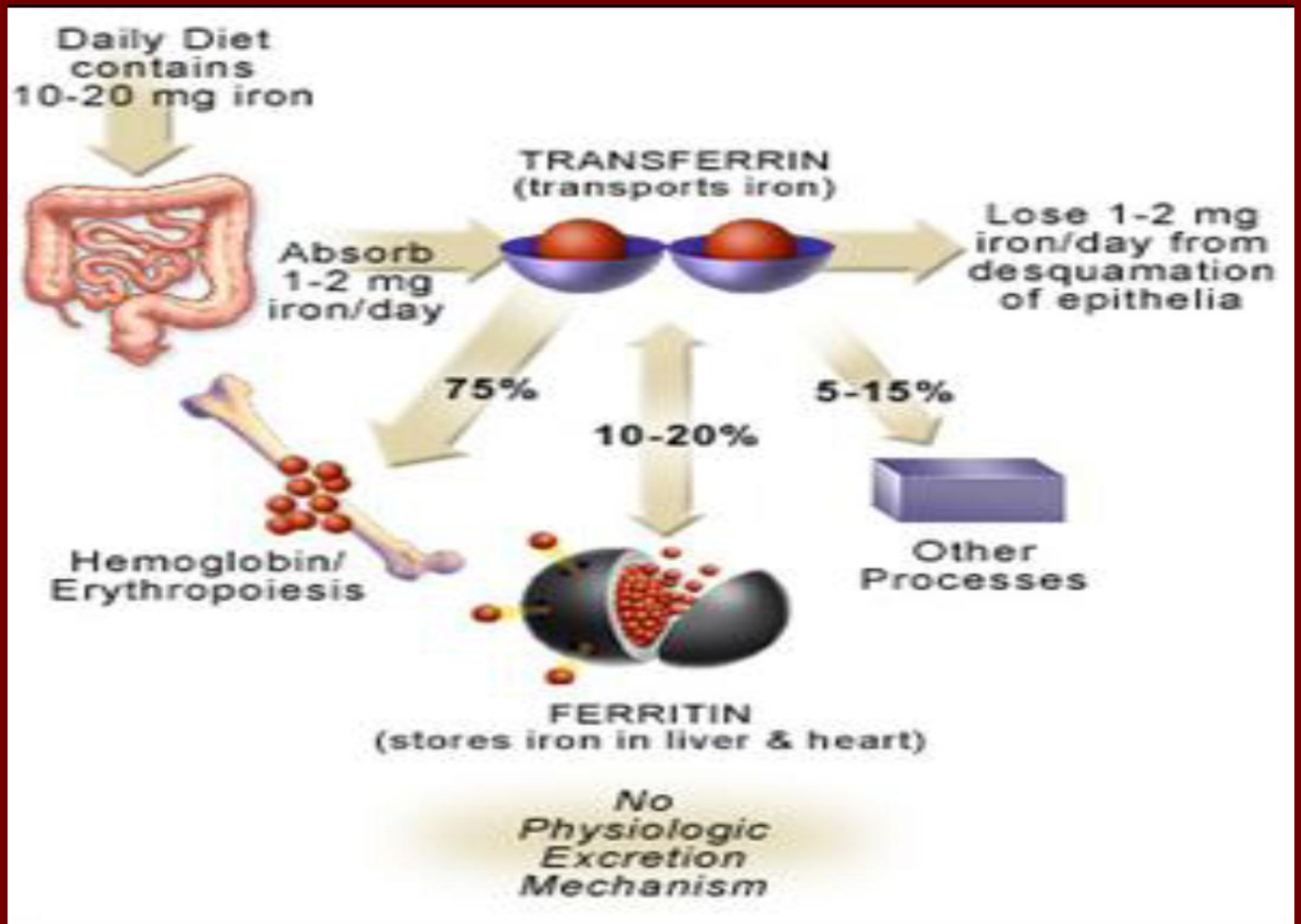
Clinical features :

Symptoms: tiredness, dizziness, fainting, lethargy.

Signs: Pallor, koilonychia (IDA), delayed capillary refilling.

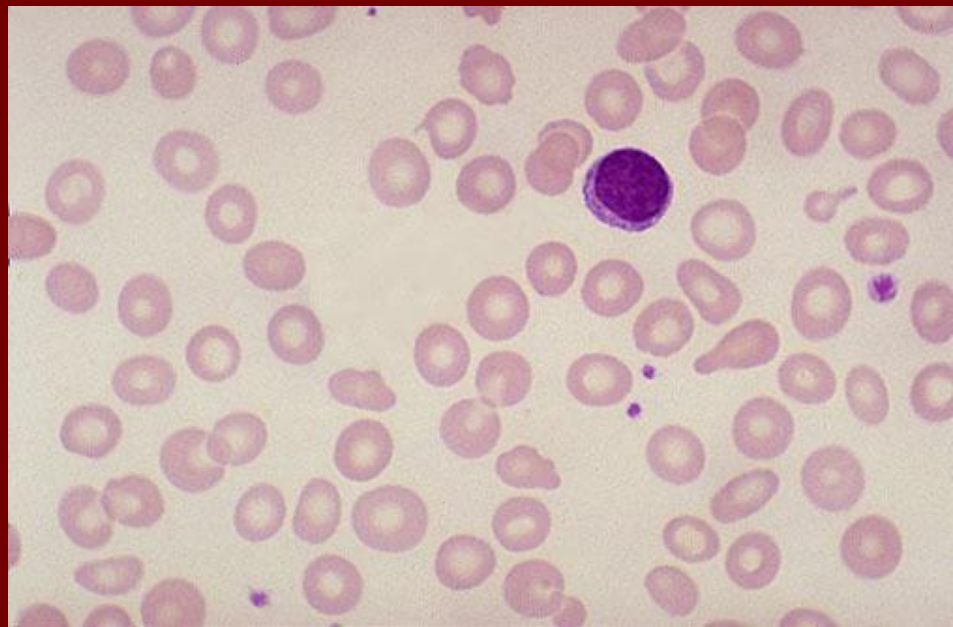
Iron deficiency anaemia (microcytic anaemia):

- Iron demand in pregnancy increases from 2 mg to 4 mg daily. A healthy diet contains 10 mg.



Investigations:

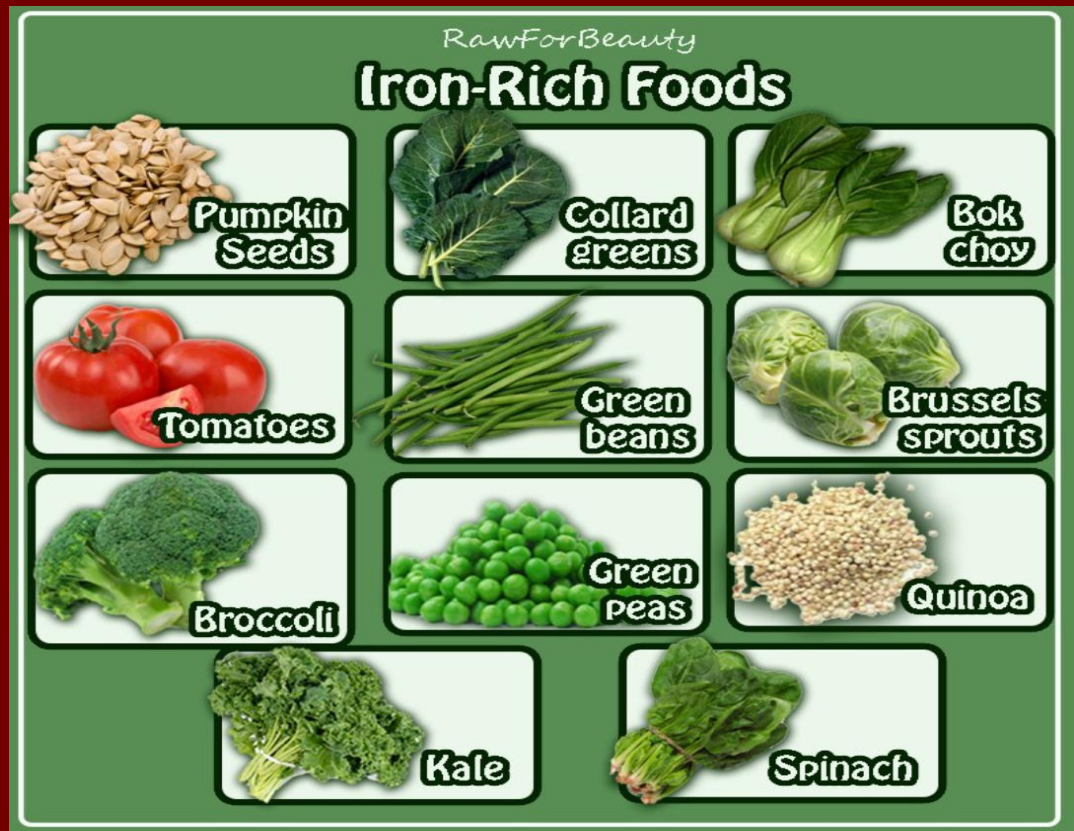
- Complete Blood count :the mean corpuscular volume (MCV) is low <85 fL.
- Blood Film: hypochromic microcytic erythrocytes
- Low levels of serum iron & ferritin with increased TIBC
- Hb electrophoresis is normal.



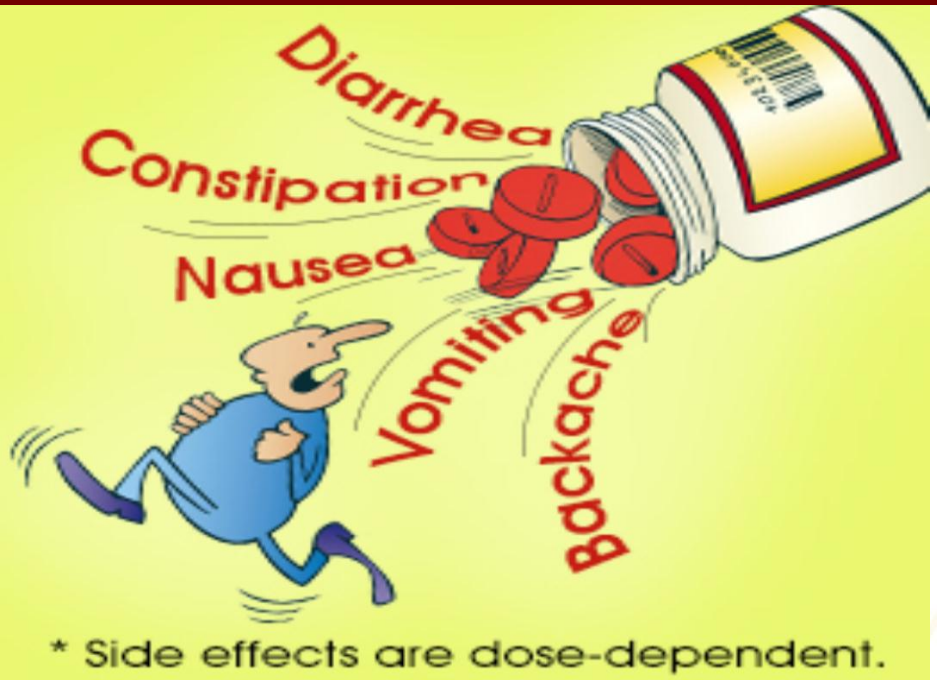
Complications:

- Impaired function of iron-dependent enzymes is the basis for the explanation of the link between iron deficiency anaemia & preterm delivery, infection, medical intervention during labour & postpartum haemorrhage.

- **Prevention:** good balanced diet & identification & treatment of iron deficiency anaemia prior to pregnancy are optimal. A policy of routine iron supplementation during pregnancy is also adopted.
- Prophylaxis: 30-60 mg elemantal iron.



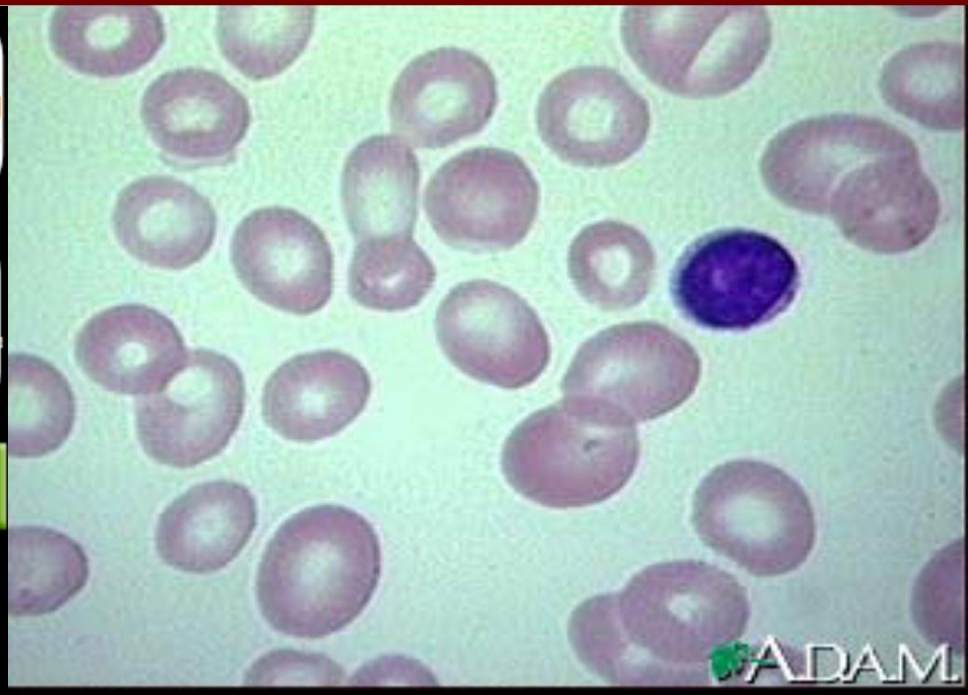
- Treatment:
- Oral iron replacement is usually effective if there is enough time (maximum increase in Hb=0.8 g/dL per week).
- the recommended dose of elemental iron is 120-200 mg daily. prophylactic doses until 3 months postpartum to ensure that iron stores are replenished.
- Therapy failure: malabsorption, loss exceed intake, poor compliance



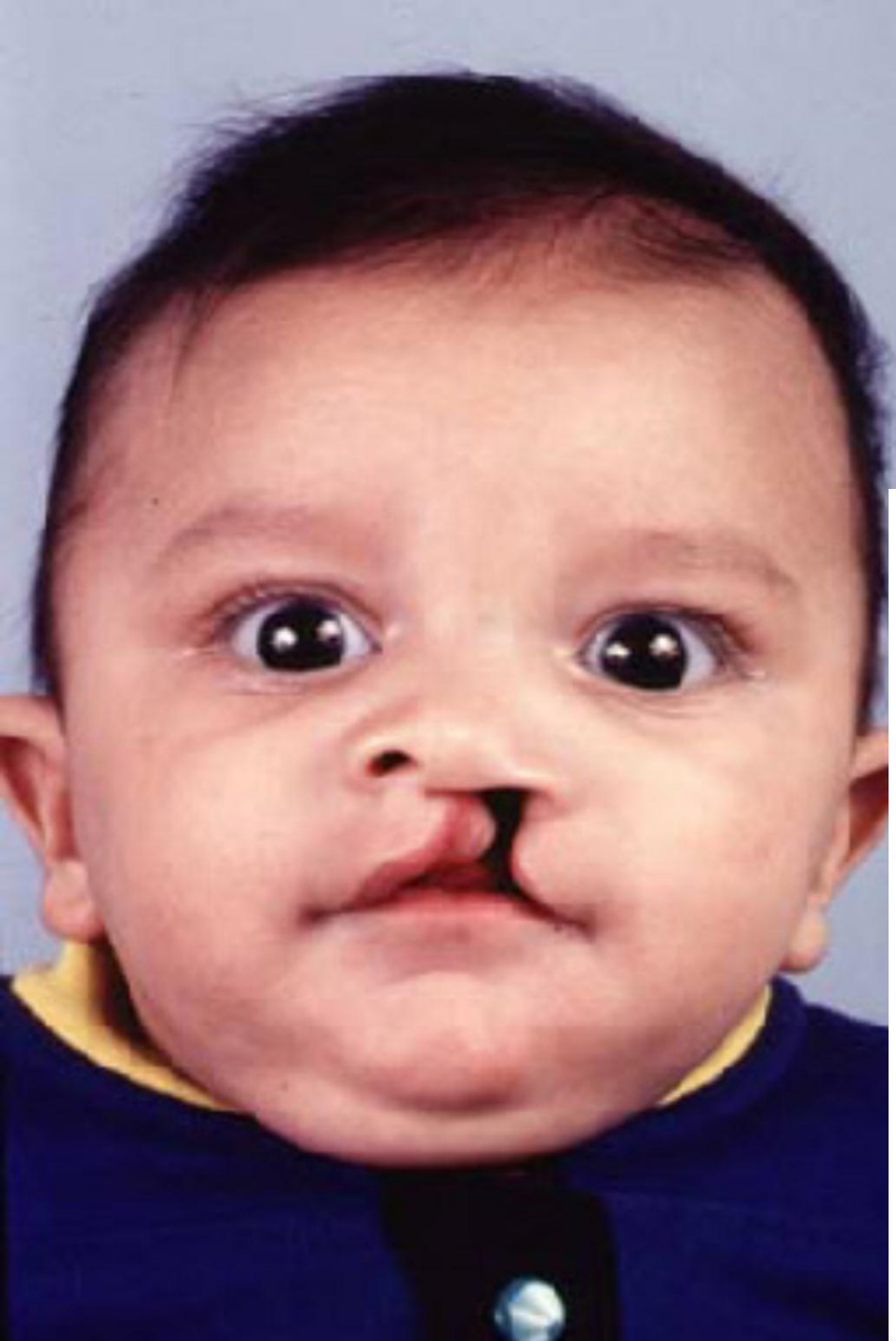
- Parenteral iron therapy for those with intolerable side effect & those with poor compliance. Response is not faster than oral iron. careful administration is required because of the possibility of anaphylaxis.
- Blood transfusion is indicated if the woman has severe anaemia beyond 36 weeks of gestation

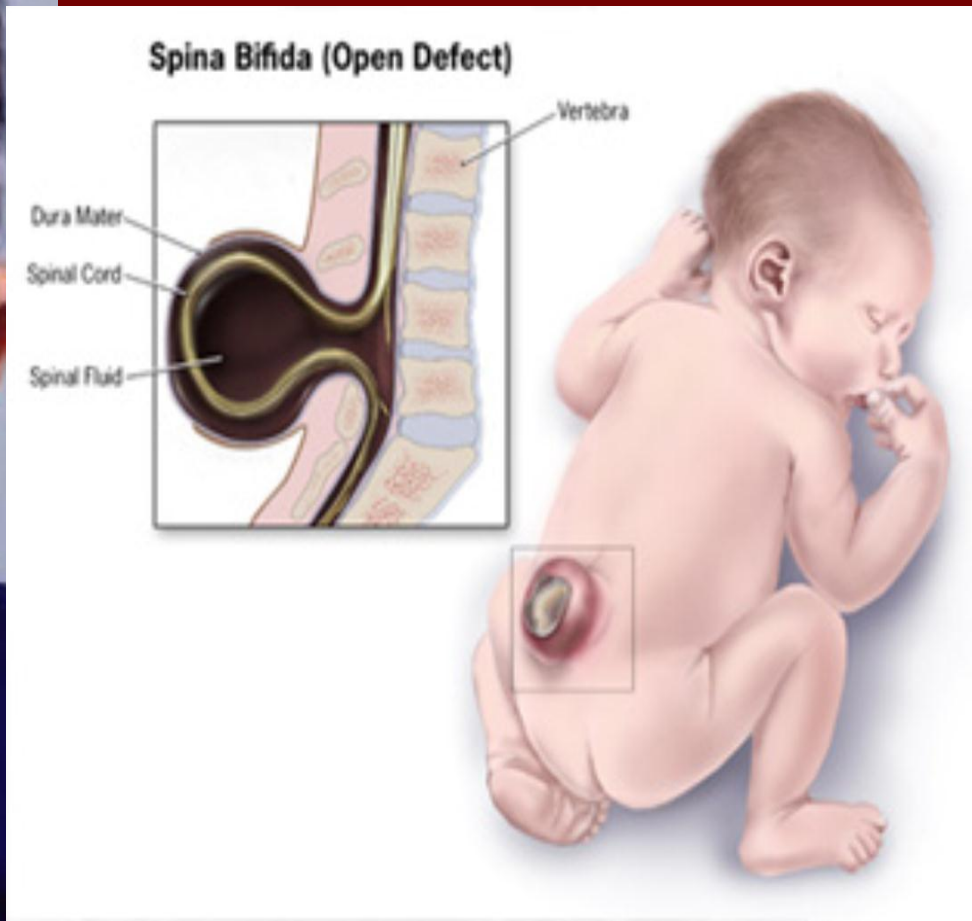
Folate deficiency

- folic acid is present in many food stuffs such as green vegetables, fruits & liver.
- increased MCV (> 100 fL) (macrocytic anaemia).
- Diagnosed by decreased red cell folate concentration



- Folate requirement is increased in pregnancy as all tissues require it for manufacture of DNA.
- Folate deficiency can occur in those with malabsorption syndrome or those with increased demand due to multiple gestation, haemolytic conditions & those taking anticonvulsant medications



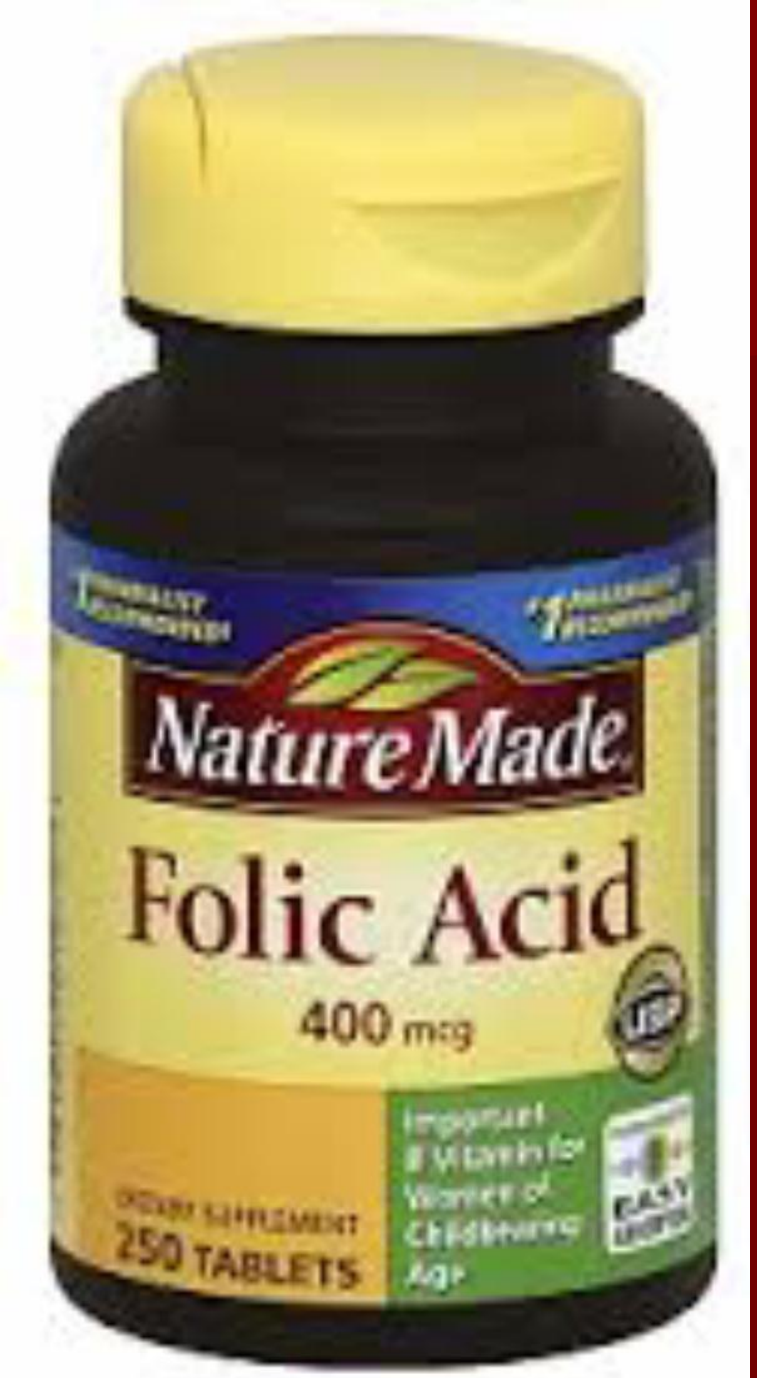

Spina Bifida (Open Defect)
Vertebra
Dura Mater
Spinal Cord
Spinal Fluid

- **Prophylaxis:** All women considering pregnancy should be encouraged to use folate supplementation (400 µg) 3 month before pregnancy , as it has been shown to reduce the incidence of neural tube defect.

  In pregnancy increase to 800 µg.

- (5mg) is required for women receiving anticonvulsant medication due to their antifolate activity or those with a history of previous child with neural tube defect & multiple pregnancy.

- **Treatment:** folic acid 5 mg daily continued for up to 4 weeks in the peurperium.


Nature Made
Folic Acid
400 mcg
250 TABLETS


actavis
Folic Acid Tablets
5 mg
28 tablets
For oral use
Folic Acid Tablets
28 tablets

Vitamin B12 deficiency:

- Causes: pernicious anemia, malabsorption, Chron's disease, tapeworm infestation
- macrocytic anemia, such cases are more likely present with infertility.
- diagnosed by increased MCV > 100 fL & decreased serum vit B12 level.
- Treatement: weekly intramuscular injection of 1000 µg B12 until anemia resolved.


Vitamin B12-
ratiopharm® N
Wirkstoff: Cyanocobalamin
Teil einer Großpackung
ratiopharm
5
Vitamin B12-
ratiopharm® N
Wirkstoff: Cyanocobalamin
Teil einer Großpackung
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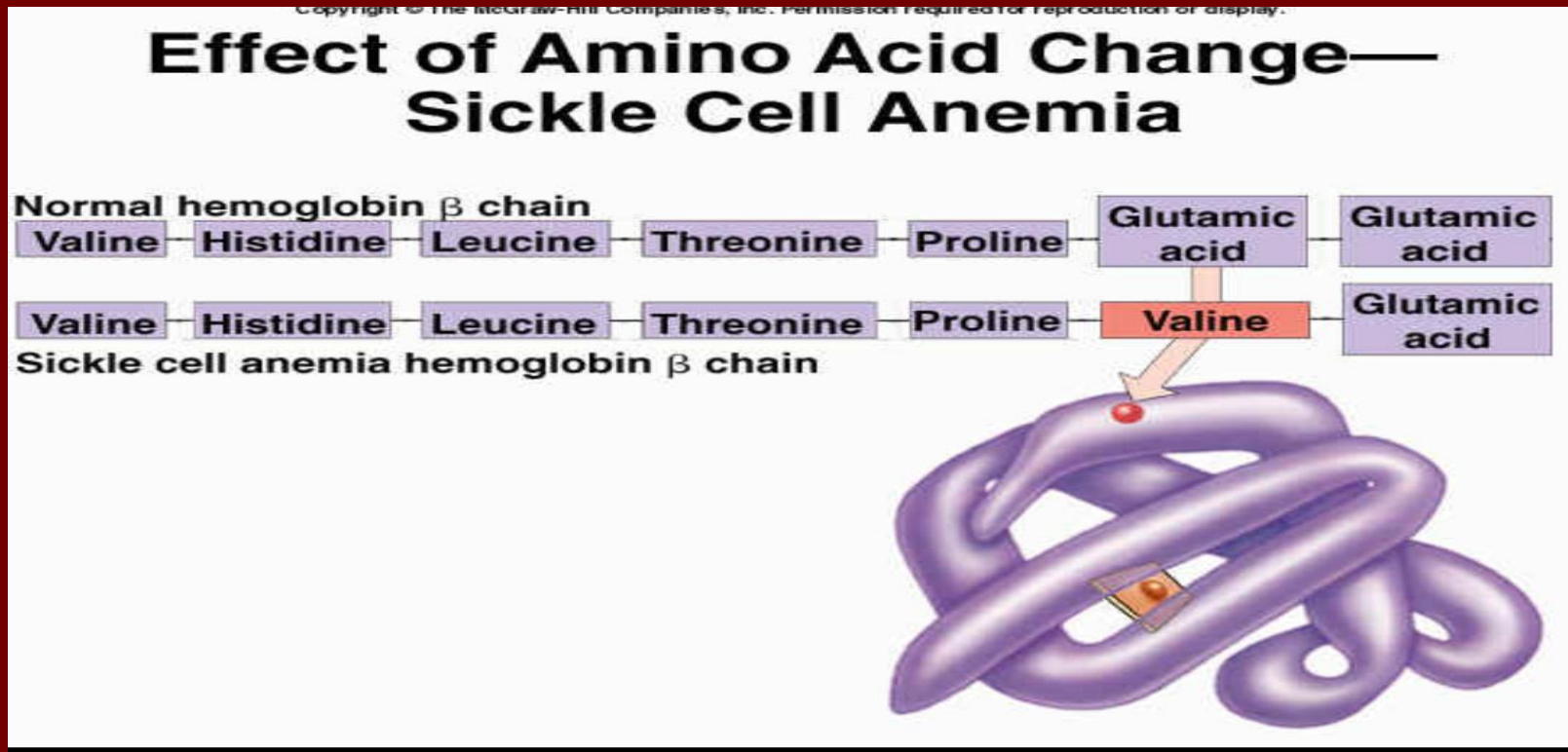
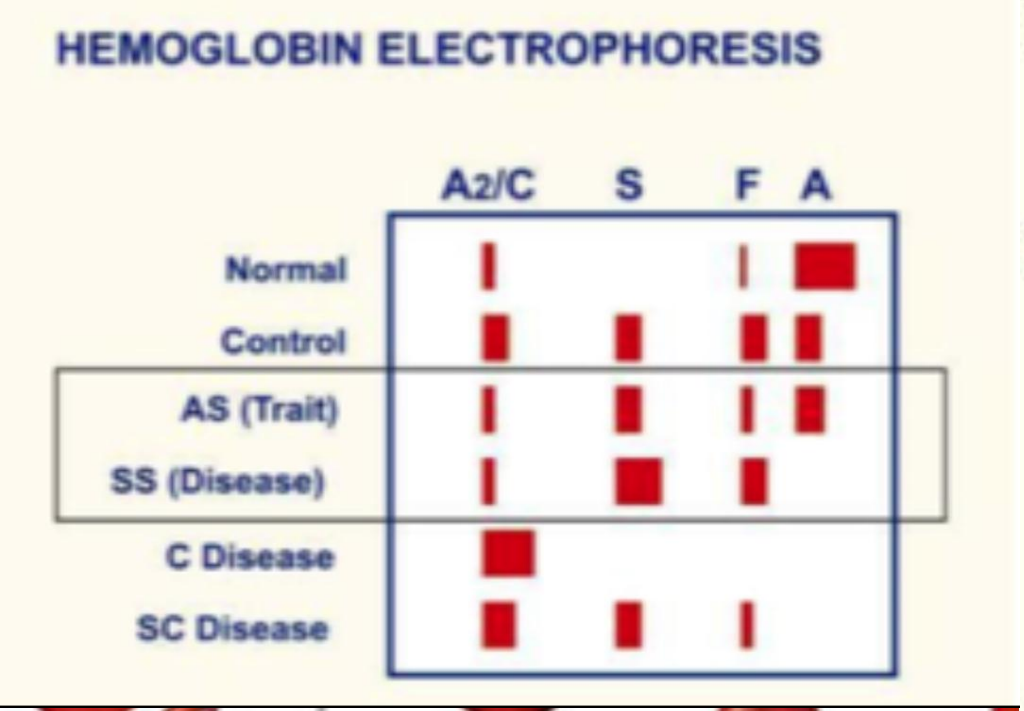
Haemoglobinopathies:

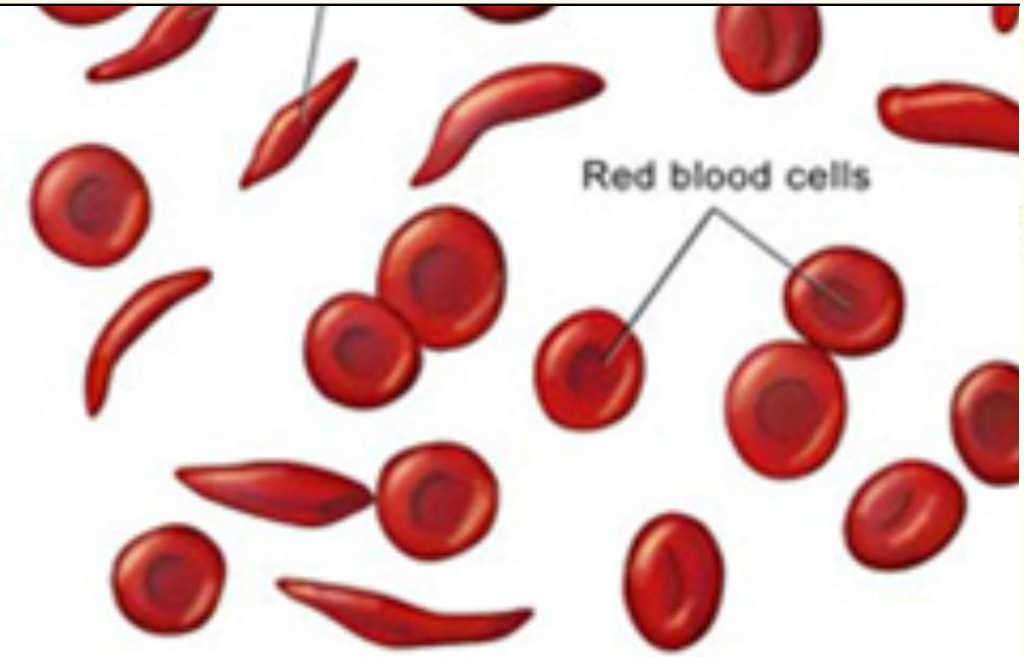
- **Sickle-cell anaemia:** an inherited disease with autosomal recessive inheritance in which abnormal haemoglobin (HbS) contains beta-chains with an amino acid substitution of valine in place of glutamine.

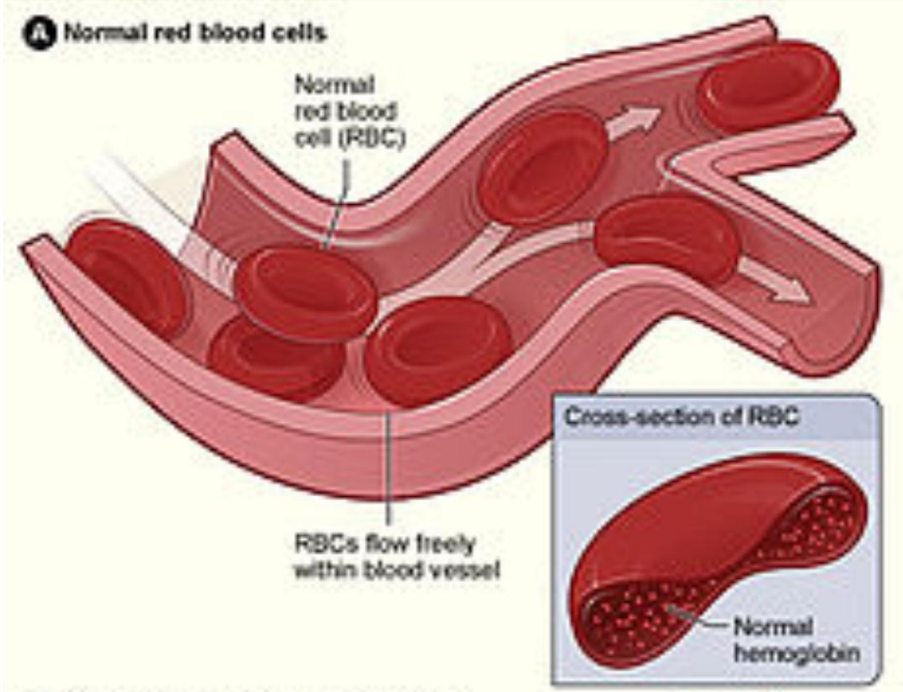


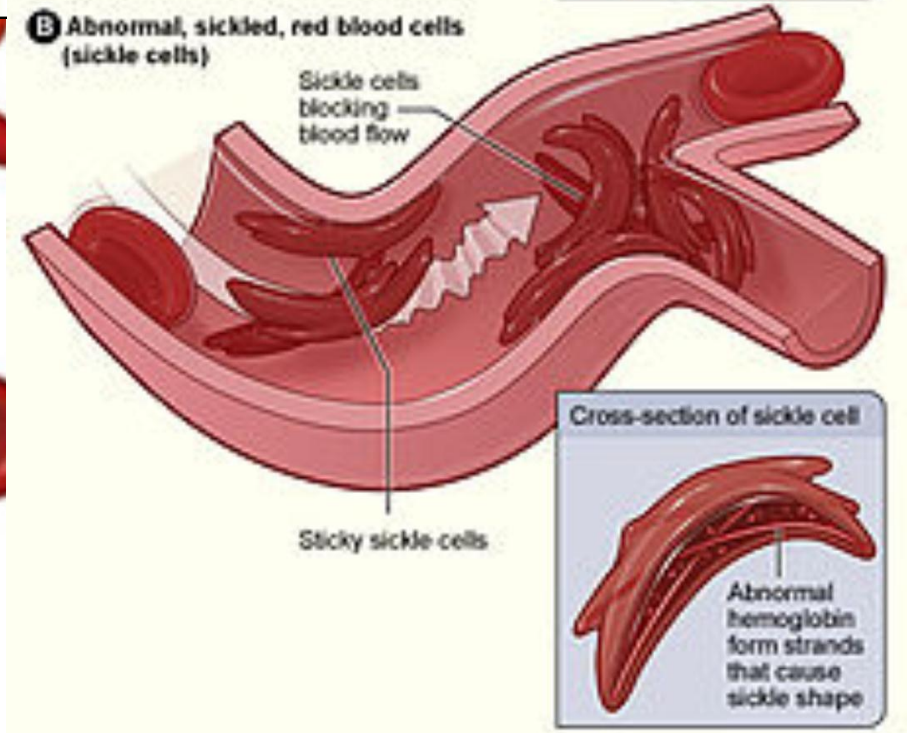
- In its deoxygenated state HbS becomes insoluble giving the red blood cells sickle shape, because of their rigid structure sickled cells block small blood vessels leading to sickling crises.

- It can be homozygous (sickle cell disease) or heterozygous (sickle cell trait).


HEMOGLOBIN ELECTROPHORESIS
A2/C
S
F
A
Normal
Control
AS (Trait)
SS (Disease)
C Disease
SC Disease


Red blood cells


A Normal red blood cells
Normal red blood cell (RBC)
RBCs flow freely within blood vessel
Cross-section of RBC
Normal hemoglobin


B Abnormal, sickled, red blood cells (sickle cells)
Sickle cells blocking blood flow
Sticky sickle cells
Cross-section of sickle cell
Abnormal hemoglobin form strands that cause sickle shape

Sickle-cell disease

- HbSS is a severe condition & in pregnancy women are at high risk of complications.
- Pregnancy is associated with increased incidence of sickle-cell crises that may result in episodes of severe pain, typically affecting the bones or lungs.
- crisis may be precipitated by hypoxia, stress, infection & haemorrhage.
- Mothers are at increased risk of miscarriage, pre-eclampsia, chest & urinary tract infection & preterm labour. The fetal loss rate is higher than normal, as is the incidence of growth restriction.

Sick-cell trait :

Carriers of the trait are usually fit & well, but are at increased risk of urinary tract infection.

Sickle-cell Haemoglobin C Disease:

may cause mild degree of anemia but is associated with very severe crises that are more common in pregnancy

Antenatal management

- Women should be screened at booking to detect haemoglobinopathies.
- No specific treatment exists to prevent sickle-cell crises;
- hypoxia, infection & dehydration should be avoided by aggressive treatment with adequate analgesia, antibiotic, oxygen & rehydration.
- Hb concentration of at least 10 g/dL with 60% normal HbA will minimize the risk of crises.
- Vaginal delivery should be the aim & epidural analgesia advised, to reduce the stress of labour.

Thalassaemia:

- The defect is a reduced production of normal haemoglobin
- The syndromes are divided into the alpha & beta types depending on which globin chain is affected.
- Normal haemoglobin consist mostly of HbA (2α 2β), with a small percentage of HbA2 (2α 2δ) & HbF (2α 2γ)

Beta-thalassaemia

- results from defects in the normal production of beta chains for which two genes are responsible
- If one of the genes for beta-chain is missing the patient will have beta-thalassaemia minor, If the two genes are missing the patient will have beta-thalassaemia major.
- These conditions can be diagnosed using haemoglobin electrophoresis.

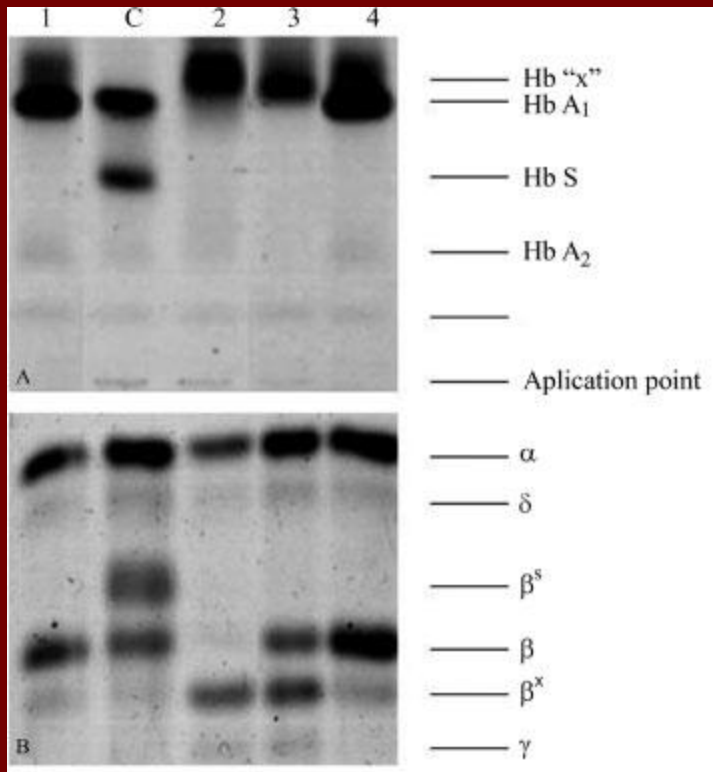


Figure 1 - Electrophoresis: (A) Alkaline electrophoresis: faster HbA migration in the propositus and his father. (B) Globin chain electrophoresis: slower β-chain migration (β^x) in the propositus and his father. Lanes: (1) mother; (2) propositus; (3) father; (4) normal brother; (C) control, heterozygous for sickle cell anemia.

- Beta-thalassaemia minor is not a problem antenatally, although women tend to be mildly anaemic with low MCV
- Oral iron & folate should be given & the partner should be screened.
- If the partner has beta-thalassaemia trait, there is 1:4 chance that the fetus has beta-thalassaemia major.
- The fetus produces HbF in utero while in postnatal life, normal HbA cannot be produced & severe anaemia develops requiring serial blood transfusion
- Eventually this leads to problems of iron overload & death.

alpha-thalassaemia minor

- there is a deletion of one, two or three of the four normal alpha genes required for haemoglobin production.
- effected individual is chronically anaemic,
- it rarely produces obstetric complications except in cases of severe blood loss

alpha-thalassaemia major

- there is no functional alpha chains,
- no normal haemoglobin is synthesized & the condition is incompatible with life
- The fetus develops marked hydrops, & pregnacies are complicated by polyhydramnios & preterm delivery.
- These pregnancies may also be complicated by severe pre-eclampsia related to the enlarged & hydropic placenta.